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The Dynamic Electrocardiogram Pattern of T Wave Inversion Following ST Segment Elevation in Acute Coronary Syndrome with Non Significant Coronary Artery Disease

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Abstract

ST segment elevation acute myocardial infarction (STEMI) is sometime indicated by typical electrocardiogram pattern, and rarely by atypical pattern. The definite diagnosis of STEMI is important to be determined rapidly and timely and becoming the key management success. The 12 lead electrocardiogram is the main diagnostic tool which should be completed and interpreted as soon as possible on patient admission. In the case, a female patient with anginal chest pain and initial ST segment elevation in electrocardiogram with non significant coronary artery disease. The subsequent electrocardiogram shows T wave inversion evolution pattern.

Keywords: STEMI; T wave inversion; variant angina; evolution

INTRODUCTION

ST segment elevation acute myocardial infarction (STEMI) is sometime indicated by typical electrocardiogram pattern, and rarely by atypical pattern. The mortality following STEMI is influenced by many factors, such as age at presentation, Killip class, delay of definitive treatment, treatment strategy, history of previous acute myocardial infarction, diabetes mellitus, renal failure, the amount of coronary artery affected, ejection fraction and therapy. The mortality during hospital admission is ranging between 6% and 14%.[1] The greatest determinant to reduce mortality rate of STEMI is the intervention strategy by primary percutaneous coronary intervention.[1]

The definite diagnosis of STEMI is important to be determined rapidly and timely and becoming the key management success, both short and long term. The 12 lead electrocardiogram is the main diagnostic tool which should be completed and interpreted as soon as possible on patient admission. The tolerable time to diagnose STEMI from electrocardiogram is 10 minute.[1] Typical electrocardiogram showing ST elevation in regional electrocardiogram leads, both precordial and extremity leads, is easily interpreted as STEMI. This quick interpretation lead to the decision to reperfusion treatment strategy, both fibrinolysis and primary percutaneous coronary intervention.

However, electrocardiogram pattern occasionally is difficult to be accurately interpreted as STEMI because of atypical pattern. Several atypical electrocardiogram pattern are bundle branch block, intraventricular conduction disturbance, ventricular pacing rhythm, isolated posterior myocardial infarction, ST elevation in aVR or non diagnostic without ST segment elevation with ongoing ischemia.[1] In these situation, electrocardiogram may not be truthfully interpreted, therefore several sequence electrocardigram recording should be performed. If possible, previous electrocardiogram should be sought to be compared with current electrocardiogram to detect the changing pattern. The diagnosis of STEMI sometimes can be precisely determined after the emergence of evolution patterns in electrocardiogram within days of hospitalisation, which is absent in the beginning of the disease.

This case report describes a case in which the diagnosis of STEMI by electrocardiogram pattern is dubious in the admission of patient with typical angina. The primary percutaneous coronary intervention shows non significant coronary artery disease, not supportive for occlusive thrombus in epicardial coronary artery. However, after several days of hospitalisation the changing infarct evolution pattern is emerged and the diagnosis of STEMI is more convincing.

CASE PRESENTATION

We report a case of a woman 56 years old whom comes to Emergency Room of Dr. Sardjito General Hospital, Yogyakarta with the chief complaint of chest pain. The pain is anginal type pain, is felt since 12 hours before hospital presentation while she swept the floor of her house. She felt heaviness on the left of her chest, piercing through her back and acommpanied with cold sweat, nausea and vomitus. She does not complaint of dyspnea or cough. She felt that the complaint worsened overtime, therefore about 12 hour later visit the privat practice medical doctor and is referred to Dr. Sardjito Hospital. The doctor says that she possibly has a heart attact.

No history of dyspnea on exertion, orthopnea or paroxismal nocturnal dyspnea. She felt previous light chest pain sometimes while doing activity and subsided while taking a rest. She had complaint of recurrent epigastric pain and sometimes accompanied with nausea and vomitus. She also had recurrent black stool. She had previous endoscopic examination for her epigastric pain and melena, and found to have gastric ulcer. The last complaint of black stool is 3 weeks previously. Patient has hypertension, but not regularly medicated. No history of diabetes mellitus or lipid disorder. She has already menopause, no history of smoking. Similar family history of chest pain or cardiac disorder is denied.

In physical examination, the general condition is good, compos mentis mental state and good nutritional status. The vital sign is blood pressure 115 mmHg/70 mmHg, pulse 88 timep per minute, respiratory rate 20 time per minute and body temperature 37 ºC. Oxygen saturation is 99%. The pain intensity perception is scored 4-5. Head examination shows no anemic conjunctiva, icteric sclerae or sianotic lip. Neck examination shows no increased jugular venous pressure or lymphadenopathy. The thoraks examination shows symmetrical movement without any abnormality in inspection. In percussion both hemithoraks are sonor. In auscultation the sound is normal vescicular sound and no basal rales. Cardiac examination shows no cardiomegaly and normal cardiac sound. Abdominal examination reveals soeffle palpaltion, normal peristaltic sound and no mass palpable. There is a minimal epigastric regional pain, The extremity examination shows warm palpattion, no edema in both leg or bluish colorisation.

The electrocardiography examination is performed, and the result shows sinus rhythm with heart rate 80 time per minute, there is notch R pattern in V3-V4 precordial leads with QRS duration 0.12 second and ST segment elevation in V3-V5 precordial leads (elevation more than 2 mm from isoelectric line). The right and posterior leads are normal in pattern. Figure 1 shows the first electrocardiogram taken in emergency room on admission.

The chest X-ray examination shows normal lung and cardiac anatomy.

The electrocardiograhy examination is repeated 20 minute later. The result of second electrocardiogram is similar to the first electrocardiogram, i.e. sinus rhythm with heart rate 80 time per minute, there is notch R pattern in V3-V4 precordial leads with QRS duration 0.12 second and ST segment elevation in V3-V5 precordial leads (elevation more than 2 mm

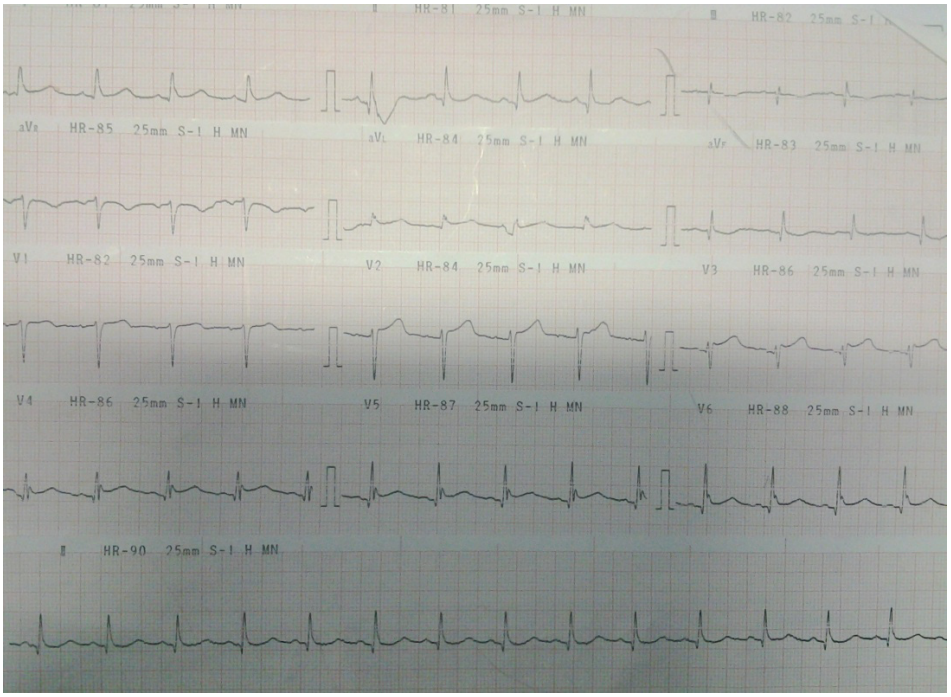

Figure 1. The first electrocardiogram on admission in emergency room. It shows sinus rhythm with heart rate 80 time per minute, R notch in V3-V5 and ST segment elevation ≥ 2 mm in V3-V5 indicate non-specific intraventricular conduction disturbance with ST segment elevation.

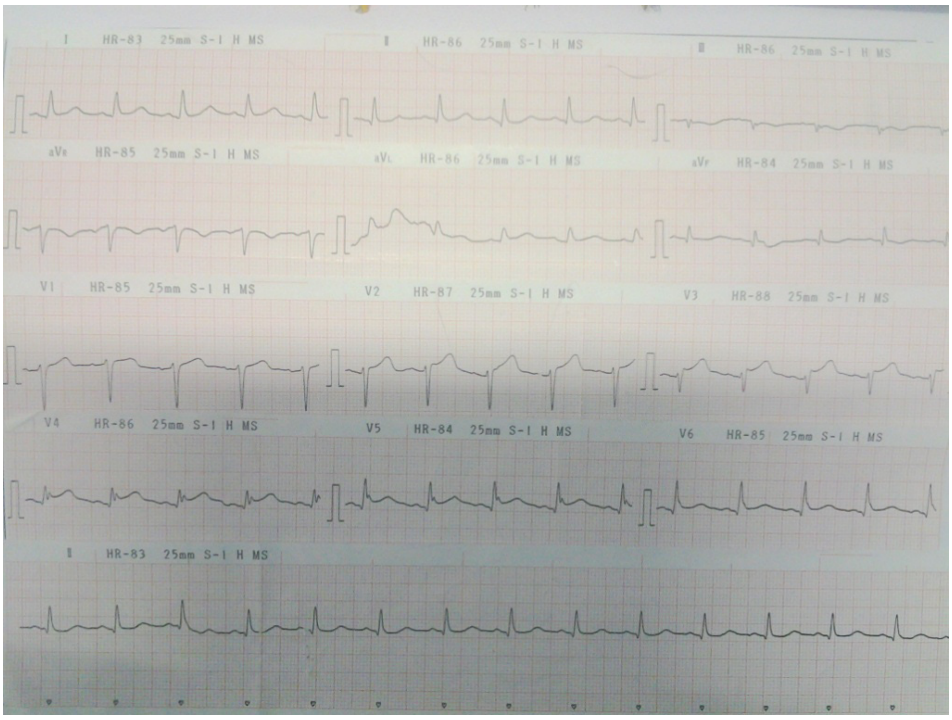

Figure 2. Electrocardiogram evaluation 20 minute form first electrocardiogram in emergency room shows similar pattern, i.e. sinus rhythm with heart rate 80 time per minute, R notch in V3-V5 and ST segment elevation ≥ 2 mm in V3-V5

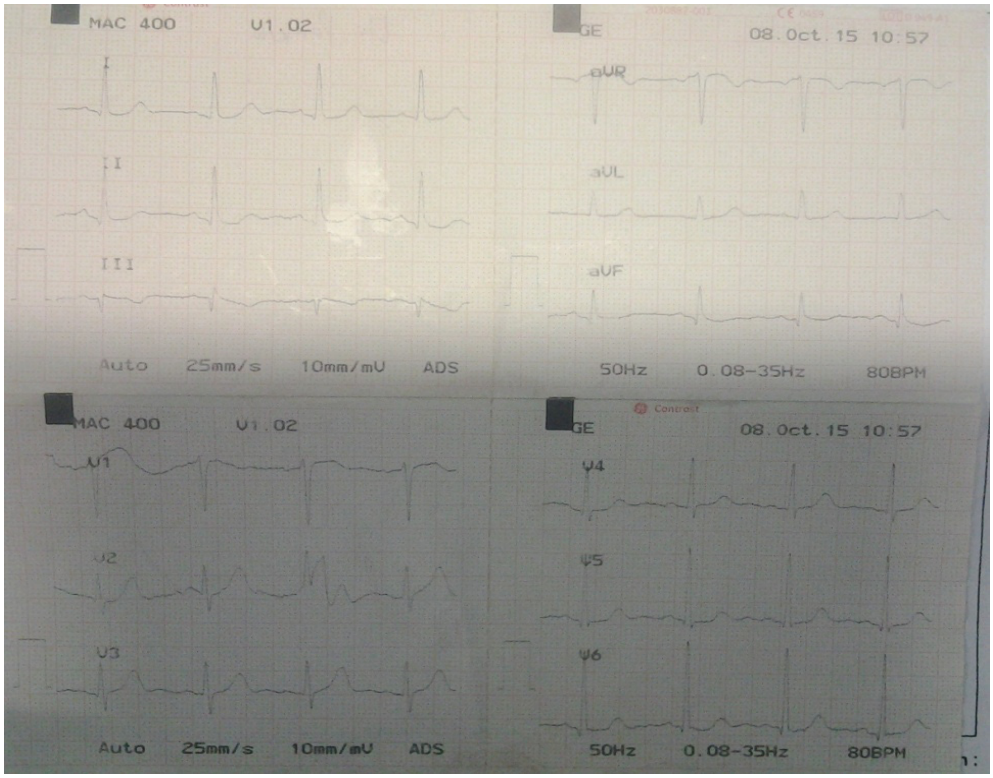

Figure 3. The electrocardiogram of the patients taken one month previously while complaining of chest pain. It shows normal sinus rhythm.

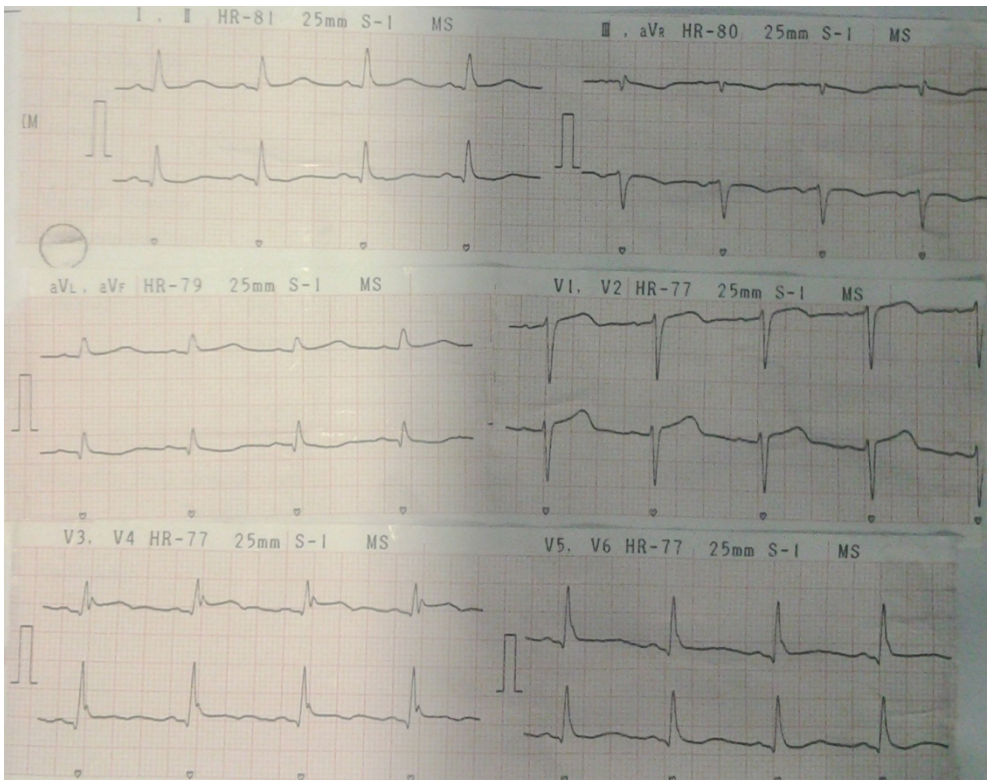

Figure 4. Electrocardiogram in the first day hospitalisation shows pada perawatan similar pattern as previously taken electrocardiogram

from isoelectric line). Figure 2 shows the second electrocardiogram, 20 minute taken after the first electrocardiogram.

The patient had previous electrocardiogram recorded 1 month previously while she visit the doctor with chest pain complaint. In the electrocardiogram shows normal sinus rhythm, different from current electrocardiogram (shown in figure 3). It indicates that there is changing electrocardiogram pattern while the patients suffered from typical chest pain. Therefore it currently supports the acute coronary syndrome.

The laboratory examination shows hemoglobin level 12.0 g/dL, leukocyte count 9.870 / mcL, platelet count 327.000 / mcL. The blood chemistry shows BUN 16.5 mg/dL, creatinine level 1.06 mg/dL, GOT level 37 mg/dl, GPT level 28 mg/dl, sodium level 145 mEq/L, kalium level 3.76 mEq/L, chlorida level 103.0 mEq/L and random glucose level 115 g/dL. Cardiac enzyme examination shows CK level 318 IU/l, CK-MB level 46 IU/l and troponin I level 1.72 mcg/l.

Based on the subjective symptom and electrocardiogram examination, the patient is diagnosed as anterior STEMI with onset > 12 hour Killip I TIMI risk score 3/14 with on going ischemia, hypertension and history of melena et causa suspicious for ulcus pepticum. With this working diagnosis, the patient is performed primary PCI.

The result of coronary angiography is normal left main, normal left anterior descendent with distal TIMI flow 2, normal left circumflex, normal right coronary artery. The conclusion of coronary angiography is non significant coronary

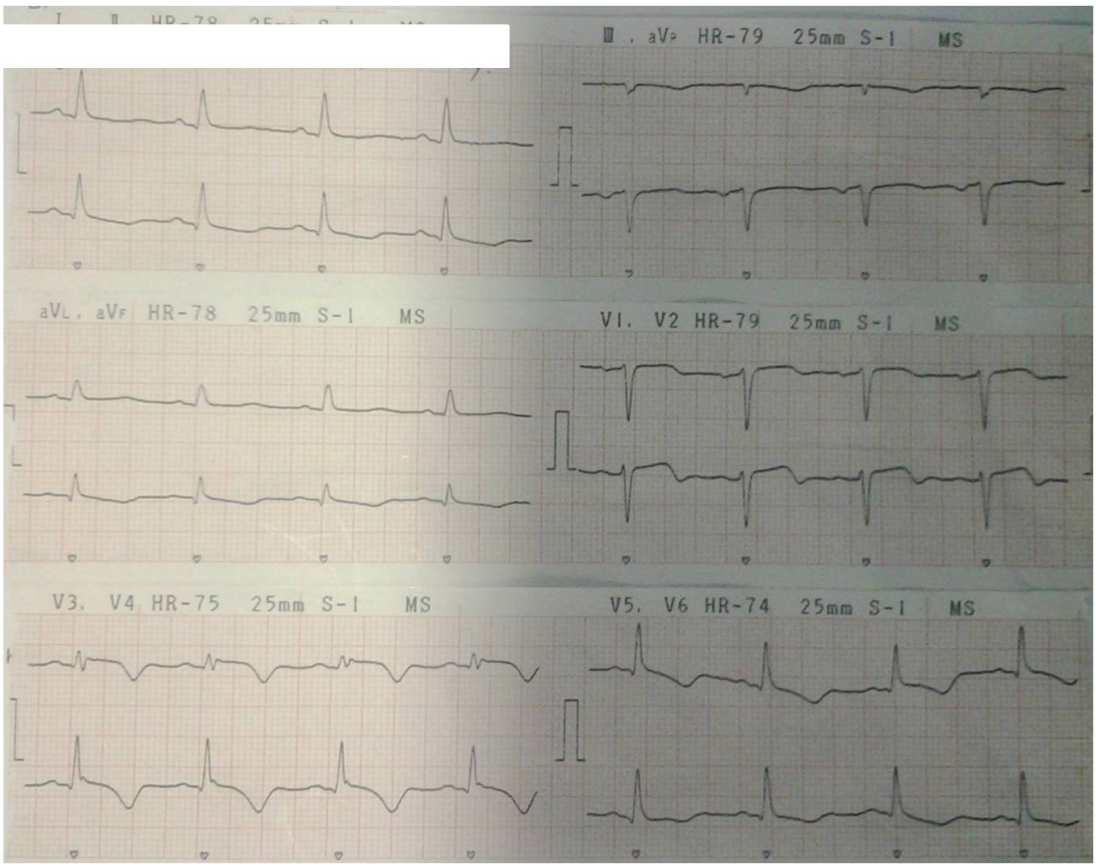

Figure 5. Electrocardiogram in the second day hospitalisation shows the changes of biphasic T wave in V1-V2 and T wave inversion in V3-V6 leads.

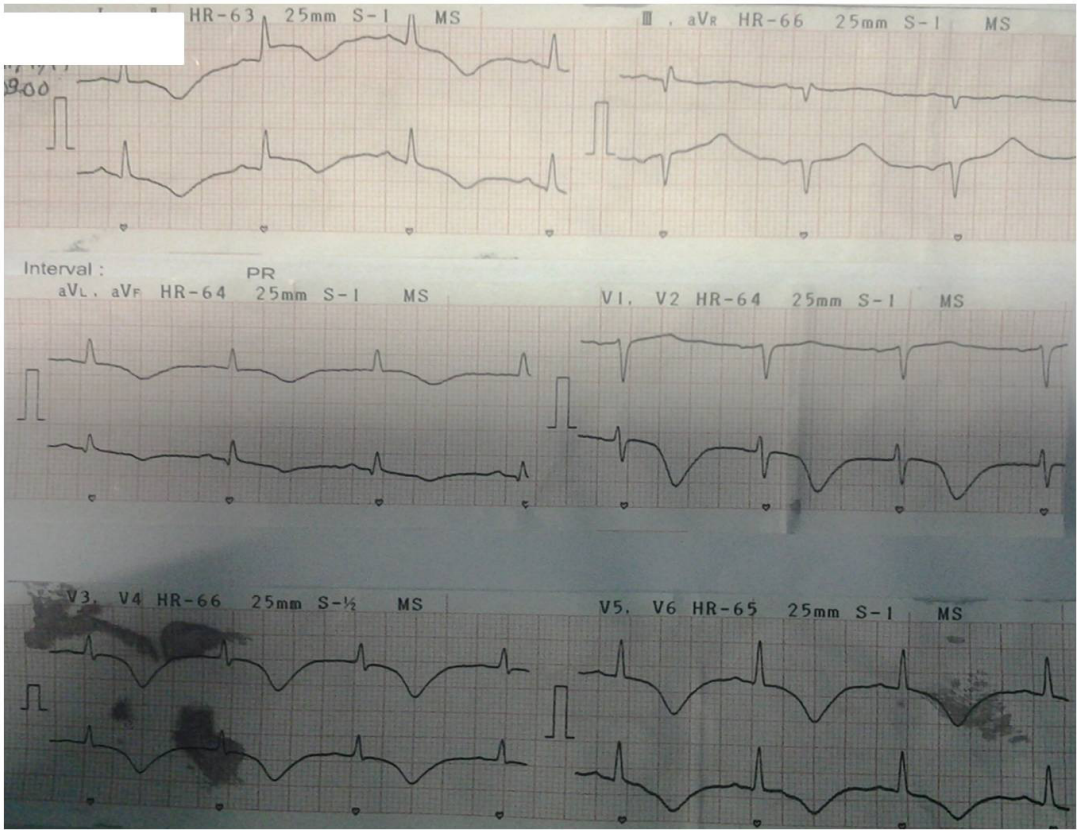

Figure 6. The electrocardiogram in the third day hospitalisation shows deeper simmetrical T wave inversion in V2-V6 leads.

artery disease. The primary PCI is not completed in this patient.

The patient is transfered into intensive cardiac care unit (ICCU) for heparinisation with unfractionated heparin, aspririn 80 mg/24 jam, clopidogrel 75 mg/24 jam, bisoprolol 2,5 mg/24 jam, captopril titrating dose and atorvastatin 40 mg/24 jam, isosorbid dinitrate if necessary.

The electrocardiogram evaluation in the first day hospitalisation shows similar resul as previous finding. Patient still complain of chest pain but with reduced intensity. Figure 4 is electrocardiogram finding in the first day.

Because the result of primary PCI shows no discernible thrombus in epicardial coronary artery and the electrocardiogram examination is no dynamic changes and evolution, the diagnostic is revised as NSTEMI with the possibility of variant angina (Prinzmetal). The cardiac enzyme evaluation shows CK level is 254 U/L, CK-MB level is 42 U/L and troponin I 0,89 mcg/L.

In the second day of hospitaisation, the chest pain complaint has subsided. In the electrocardiogram evaluation, there are changes in anterior precordial leads that indicate evolution pattern on T wave, i.e. T inversion pattern in V2-V5 leads. There is no detectable Q wave in the electrocardiogram (figure 5).

In the third day hospitalisation, the chest pain is diminished. Treatment given at the time are *unfractionated heparin*, aspirin 80 mg/24 h, clopidogrel 75 mg/24 h, bisoprolol 2.5 mg/24 h, captopril 25 mg/ 8 h and atorvastatin 40 mg/24 h. From the electrocardiogram evaluation, the T wave inversion in V2-V6 leads is getting deeper. The T wave inversion is also detected in inferior leads (III dan aVF) (figure 6).

Because of the changes of electrocardiogram and the initial electrocardiogram shows ST segment elevation, the final diagnosis is anterior STEMI. In the subsequent hospitalisation, the chest pain is diminished, electrocardiogram shows non Q wave evolution pattern with T wave inversion in anterior and inferior leads. The next follow up is uneventful and the patient is discharged home in day 6 hospitalisation.

DISCUSSION

We have reported a female patient, 56 years of age, with chief complaint of anginal chest pain and the sign of ST segment elevation in the anterior lead in the electrocardiogram. The coronary angiography reveals non significant coronary artery disease. In the course of hospitalisation, the electrocardiogram undergoing dynamic changes in the pattern of non Q wave evolution, i.e. T wave inversion in the anterior precordial leads in the leads which previously showed ST segment elevation. The final diagnosis of this patient is anterior STEMI.

The electrocardiography examination is a main initial clinical examination to diagnose myocardial ischemia or infarction. The correct interpretation, especially in the emergency setting, is a base to decide the prompt intervention and/or the need to perform other diagnostic modality. The early changes in electrocardiogram which indicate acute myocardial ischemia or infarct are peak T wave or hyperacute T, ST segment elevation and/or depression, QRS complex changes and T wave inversion. The changes in ST segment is due to the flow of current which is known as injury current.[2] The injury current is a result of voltage gradient accross ischemic myocardial and non ischemic myocardial during resting phase and plateau phase of ventricular action potential.[2] The changes in this phase is associated with TQ segment and ST segment in the electrocardiogram.[2]

Several factors involve and influence in the electrocardiogram pattern in patients with acute myocardial infarction, the most common mechanism are : (1) the disturbance in the level of cells due to myocardial ischemia resulting from coronary artery total occlusion or subtotal occlusion with or without distal embolisation, (2) duration of ischemia process, (3) myoardial ischemia extention : transmural versus subendocardial, (4) the degree of ischemia which is influenced by residual flow due to collateral or antegrade flow and the presence of ischemic or pharmacologic preconditioning, (5) localisation in relation with electrocardiogram leads, (6) underlying abnormalities, such as interventricular conduction disturbance, left ventricular hypertrophy, primary or secondary repolarisation abnormality, cardiac pacemaker implantation, and (7) coronary artery anatomical variation.[3]

In the patient with myocardial ischemia due to reduced blood supply, the early pattern of 12-lead electrocardiogram can be two patterns, i.e. (1) predominant ST segment elevation as a sign of ST elevation acute coronary syndrome and (2) no predominant ST segment elevation, therefore patients is classified as non ST elevation acute coronary syndrome.[3] The first sign may be classified as aborted myocardial infarction or persistent STEMI based on the detectable myocardial necrosis biomarkers in the blood. Aborted myocardial ischemia may be due to spontaneous reperfusion or reperfusion due to intervention before proceeding into myocardial necrosis.[3]

The changes in QRS complex reflect alteration in electrical activation in the region undergoing ischemia or infarction. The severity and extent of electrocardiogram changes depend upon the size and location of ischemic or infarct region.[2] The size and location of affected region depend upon the involvement of coronary artery, the location of coronary artery occlusion and the presence of collateral circulation.[2]

The pattern of electrocardiogram in the acute phase and its evolution during acute coronary syndrome can be classified in the two pathologic categories, i.e. transmural and subendocardial ischemia. In the electrocardiogram pattern, two pattern indicate transmural ischemia: ST segment elevation and non ST segment elevation. The ST segment elevation pattern can be divided into two patterns : typical ST-ACS pattern and STEMI equivalent : mirror image pattern. The non ST segment elevation pattern can be two patterns, i.e. hyperacute T: peak tall T wave and post acute phase: deep symmetrical T wave inversion in precordial leads.[3]

The electrocardiogram pattern which indicate subendocardial ischemia are classifies as three different patterns: (1) circumferential ST segment depression, (2) regional ST segment depression, and (3) T wave inversion.[3] In patients with NSTE-ACS, normal electrocardiogram is detected in 15-20 % patients. As many as 25% of patients with NSTE-ACS, the electrocardiogram pattern can be difficult to interpret due to the presence of confounding such as bundle branch block, left ventricle hypertrophy, Wolff-Parkinson-White pattern or pacing rhythm.[3]

The typical patterns of electrocardiogram in STEMI undergoing evolutional changes are: (1) hyperacute T wave, (2) ST segment elevation, (3) pathological/abnormal Q wave, (4) T wave inversion and (5) ST segment normalisation.[3] Inverted T wave as a part of STEMI evolution is due to ischemia in the epicardium. T wave inversion is suggested because of delayed depolarisation in the ischemic tissue. In normal heart, epicardium is the first region undergoing depolarisation, whereas the endocardium is the last. The delayed repolarisation in epicardium during ischemia will change the direction of ventricular repolarisation.[4] The repolarisation current from endocardium to epicardium makes repolarisation vector inversed and causes T wave negative deflection. T wave inversion occurs in about ¾ patients suffer from complete myocardial necrosis. The presence of T wave inversion in precordial leads indicate the 86% positive predictive value of LAD stenosis.[4]

T wave evolution in STEMI is not a sign of cell death, rather it indicates the alteration in ion channels in viable myocardial area after episode of severe ischemia. In patients who undergo T wave inversion, reischemic episode often correlate with alteration in T wave vector with positive T wave with or without ST segment elevation (pseudonormalisation) in the ischemic area.[3] Early T wave inversion accompanied by ST segment resolution is a sign of reperfusion. However, the significance of negative T wave in the lead with previous ST segment elevation before reperfusion therapy is unknown. The presence of T wave inversion in previously ST segment elevation leads indicate the myocardial residual perfusion due to spontaneous recanalisation from infarct-related artery or the presence of collateral.[5] Collateral vessels can develop especially in patients with chronic coronary artery severe stenosis.[5]

T wave inversion can occur in the post acute phase of ACS. The pattern is reflected by deep and symmetrical T wave inversion in precordial leads. These patterns occur in 20% patients diagnose with ACS and undergo impending acute myocardial ischemia.[3] This pattern consist of minimal ST segment elevation or isoelectric ST segment and T wave inversion in precordial leads. In this pattern, usually there is no discernible changes in QRS complex. The electrocardiogram pattern can persist until 24 hours. In patients with this pattern, coronary angiography reveals significant stenosis in proximal LAD. More than 50% patients undergo subtotal stenosis and 1/5 is totally occluded with the presence of collateral vessels in the LAD teritory.[3] T wave inversion with Q wave is thought

to be natural evolution of STEMI that occurs in patients wihtout reperfusion. Some investigation show the appearance of T wave after reperfusion is a sign of patent infarct-related artery.[6]

Clinical implication of electrocardiogram pattern as T wave inversion with minmal ST segment elevation or isoelectric ST segment in V1/V2 – V3/V4 leads and clinical suspicion of acute coronary syndrome is an indication of post ischemic in anterior wall due to significant stenosis of proximal part of LAD. The infarct-related artery has been reopened or adequate collateral sirkulation. In patients undergoing fibrinolysis or percutaneous coronary intervention, this particular electrocardiogram pattern.[3]

In this case, there is no significant identifiable culprit artery or thrombotic lesion from coronary angiography. The other possibility is previous reperfusion or the presence of variant angina (Prinzmetal). Clinically, the hallmark of variant angina is anginal episode in resting and recurrence which is associated with transient ST segment elevation. The use of short acting nitrate will relieve the angina. The coronary angiography in variant angina usually shows normal coronary or non significant stenosis. Only a few patients show significant coronary artery stenosis overlapping with coronary spasme.[7] The key to diagnose variant angina is documented ST segment elevation during anginal chest pain (at rest) and the elevation back to normal/isoelectric when the pain subsided.[8] The case has history of recurrent chest pain. The possibility of variant angina can not be excluded, with the current episode is progressing to anterior STEMI.

CONCLUSION

We have reported a female patient, 56 years old, with anginal chest pain and the changing electrocardiogram pattern as non specific intraventricular conduction disturbance with typical ST segment elevation. The coronary angiography shows non significant corornary artery disease. The subsequent electrocardiogram pattern is changing as evolution type symmetrical T wave inversion in anterior leads in the previous ST segment elevation. The diagnosis of the case is anterior STEMI with the possibility of variant angina (Prinzmetal) which progress to STEMI anterior.

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